

CASE REPORT



Left anterior descending artery hyper dominance giving rise to the posterior descending artery: an extremely rare coronary anomaly and its clinical implications

Muhammad Shabbir Rawala ^{a,b}, Alex Munoz^c, S. Tahira Shah Naqvi ^d and Mohammad Hassan Pervaiz^a

^aDepartment of Medicine, WVU-Charleston Division, Charleston, WV, USA; ^bDepartment of Medicine, Rapides Regional Medical Center, Alexandria, LA, USA; ^cDepartment of Radiology, University of Kentucky, Lexington, KY, USA; ^dDepartment of Medicine, Jinnah Medical & Dental College, Karachi, Pakistan

ABSTRACT

The prevalence of congenital coronary artery anomalies is approximately 1% in the general population. They are a common cause of sudden death in younger persons. The origination of the posterior descending artery (PDA) from left anterior descending (LAD) artery is an extremely rare anomaly. We present a case of a 54-year-old female who presented with diabetic ketoacidosis with co-existing non-ST elevation myocardial infarction, therefore, had an invasive angiogram that identified the anomalous origin of PDA from LAD. It is vital to define coronary anatomy as anomalies dictate which cardiac intervention should be attempted in cases of ischemia.

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Coronary artery anomaly; left anterior descending artery; posterior descending artery

Introduction

Anomalies of the coronary arteries are usually an incidental finding in 0.3% to 1% of the healthy individuals [1]. Based on what their potential is in causing myocardial ischemia, they are classified as benign or malignant [2].

Recognition of the various types of anomalies on angiography may be difficult and failing to recognize them may lead to prolongation of the angiographic procedure or may result in repeated catheterizations. Correct identification of the course of coronary arteries is essential for the anatomic classification on the angiogram as well as for revascularization bypass surgery patients [3]. In approximately 85% of people the coronary circulation is right dominant with the posterior descending artery (PDA) arising from the right coronary artery (RCA). In only about 10–15% of cases, PDA arises from the circumflex artery (LCX), or even less commonly from both RCA and LCX (7%) [4,5]. Very rarely PDA can be seen to arise from the left anterior descending (LAD) artery. In this case report, we will discuss an extremely rare anomaly where the LAD runs across the left ventricular apex and gives rise to the PDA, which terminates beyond the crux.

Case Presentation

A 54-year-old female presented to the emergency department (ED) with nausea, vomiting and altered mental status. She had a prior medical history of type

II diabetes mellitus and hypertension. The history at the time of admission was limited due to patient having altered mental status, however, later when her mental status came back to baseline she did not report any cardiac history nor any episodes of angina or chest pain. She denied any history of tobacco or drug use. On examination, her vitals were blood pressure 95/50 mm Hg, pulse 116, respiratory rate of 14/minute on ventilator, temperature of 98.2 F. Her general appearance was lethargic, obtunded, altered mental status, and on cardiac examination, S1 and S2 were audible without any gallop or murmur, no JVD or edema appreciated. On further evaluation, she was found to have diabetic ketoacidosis with severe metabolic acidosis and a pH of 6.8. The EKG showed sinus tachycardia and minimal ST depression in leads V3–V4. On laboratory workup, her CK-MB was 7.1 ng/ml (reference range 0.0–4.3 ng/ml) along with an elevation in troponins at 2.27 ng/ml (reference range 0.0–0.1 ng/ml) signifying non-ST elevation myocardial infarction (NSTEMI). The patient was admitted in ICU as she had been intubated in ED due to altered sensorium and severe metabolic acidosis. She was started on intravenous fluid, sodium bicarbonate, and empiric antibiotics. In the subsequent days, the patient was successfully extubated; however, her CK-MB and troponin continued to trend up to 26.8 ng/ml and 6.7 ng/ml respectively. It was initially believed that patient was having Type-II NSTEMI due to diabetic ketoacidosis. After stabilization of the patient's condition, an elective left

CONTACT Muhammad Shabbir Rawala  muhammad_rawala@hotmail.com  Department of Medicine, Rapides Regional Medical Center, 6509 Coliseum Blvd, Unit 10, Alexandria, LA 71303

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heart catheterization (LHC) was performed for evaluation of NSTEMI. The LHC revealed a LAD that was of large caliber and after giving off diagonal branches wrapped around the apex to give rise to the PDA that terminated beyond the crux of the heart. (Figure 1-II). The LHC did not show any obstructive coronary artery disease but identified a non-dominant RCA and LCx. (Figure 3)

Discussion

The coronary vasculature is meticulously regulated by a series of transient events which include vasculogenesis, angiogenesis, arteriogenesis, and remodeling. The embryonic epicardium and the coronary vascular system constitute as a single development unit. In early stages, myocardium forms the outer most layer of the

embryonic heart, and at this point, coronary vessels do not form. The epicardial formation is therefore seen to be necessary for the formation of the epicardial vessels. The spread of the primitive epicardium over the myocardium is considered as the mark when the coronary vascularization begins [6].

The epicardium is derived primarily from the proepicardial serosa, which is the extracardiac primordium. The proepicardial serosa and the epicardium are then believed to give rise to almost all the cellular elements of the intramyocardial and subepicardial connective tissue, and also to all of the coronary vasculatures [7].

This is a rather complicated and not well-understood process that requires more research.

It has been shown in some experiments that disrupting myocardial cell polarity can influence the

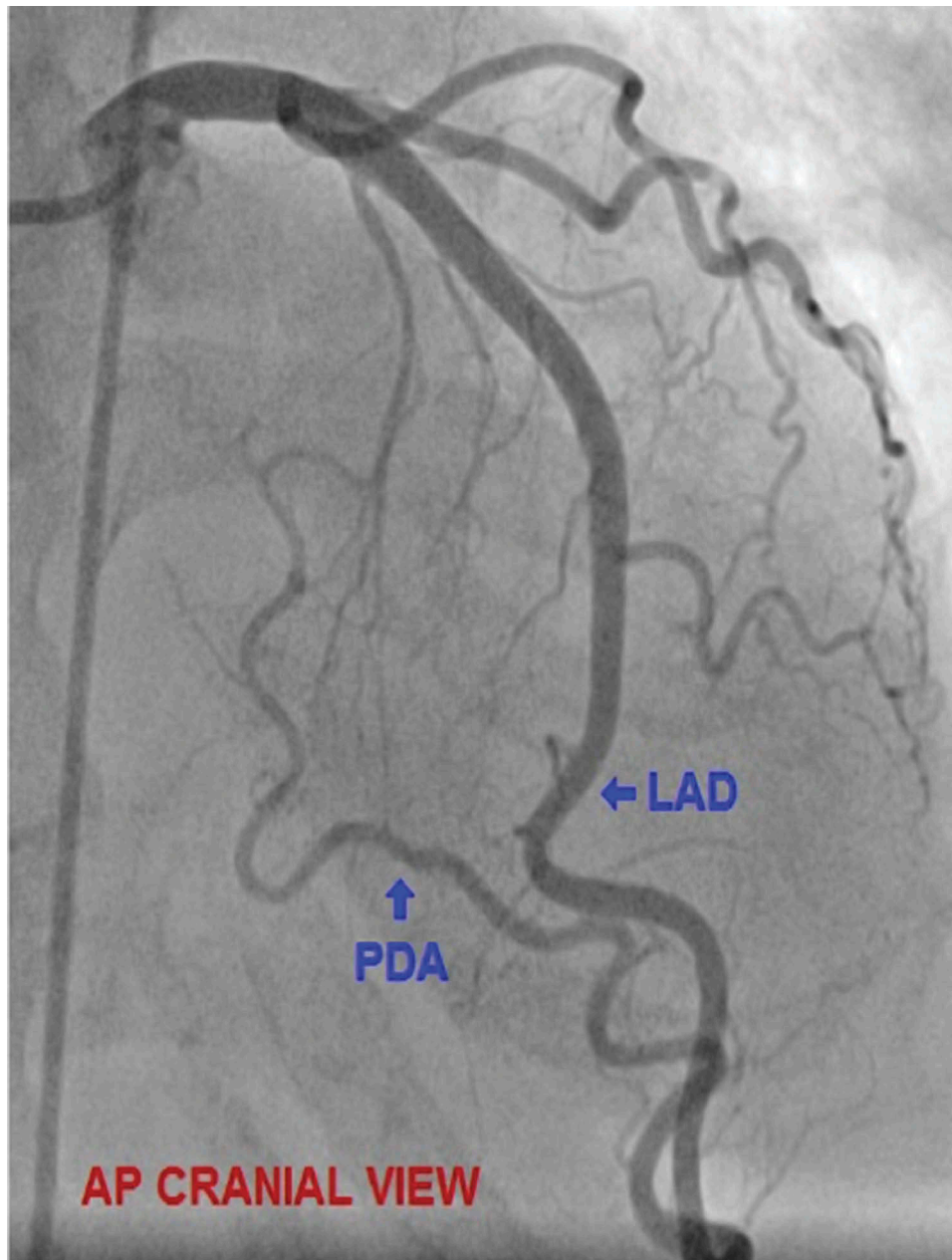


Figure 1. The left anterior descending artery giving off the posterior descending artery.

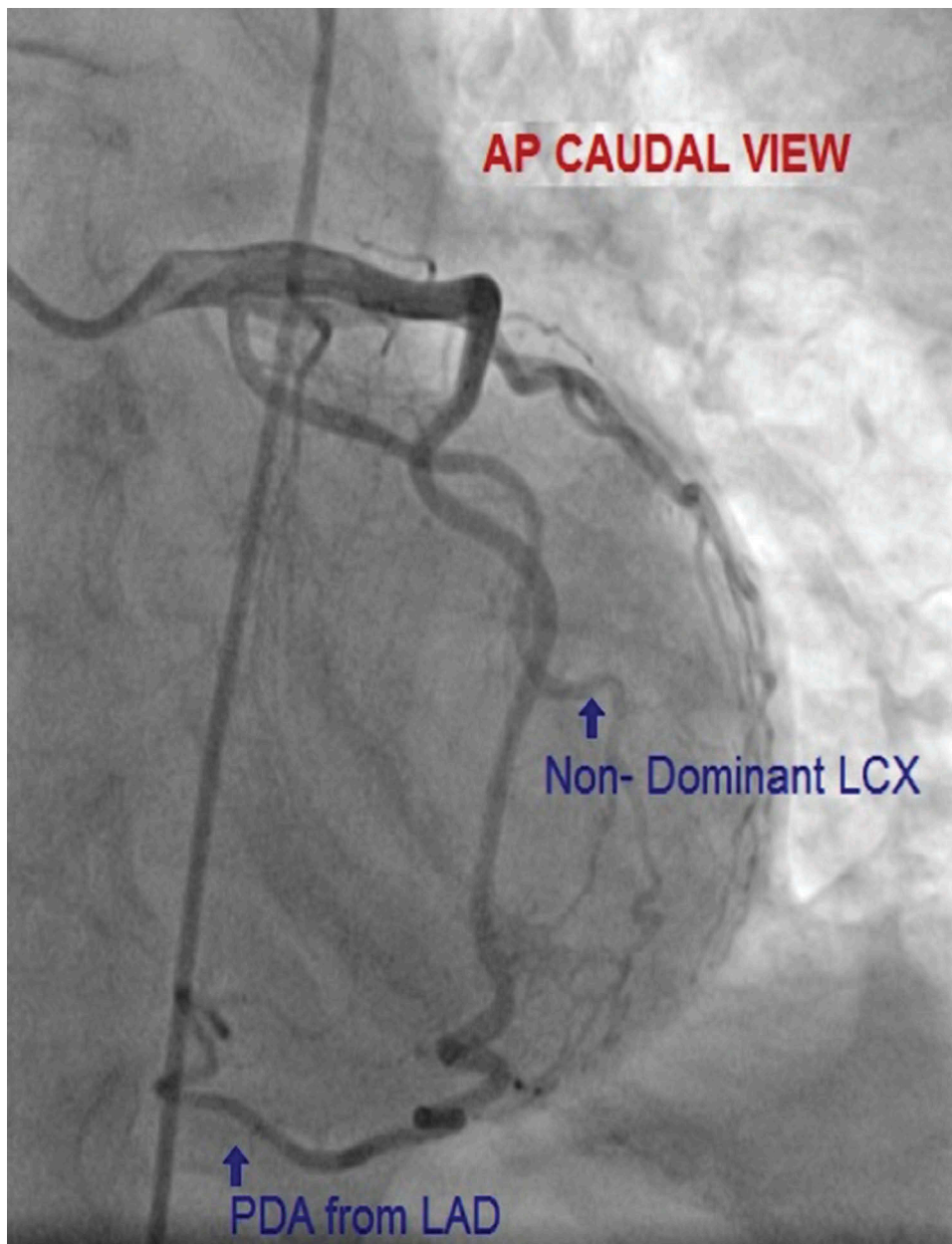


Figure 2. Invasive angiogram showing the circumflex artery and the left anterior descending artery giving off the posterior descending artery.

patterning and mural location of coronary arteries. Although the mechanism remains unknown, it is presumed that embryonic coronary arteries grow towards the aortic root from the ventricles, eventually establishing the systemic blood flow through the coronary ostia [8–10]. Very little explanation is currently available that would shed light on the anomalous formation of the coronary circulation.

The dominance of coronary circulation is determined by whether the PDA arises from the RCA or the left coronary artery. On angiography, Type III LAD is commonly seen. However, when it continues over the apex of the heart to the posterior interventricular groove as the PDA, it is described as ‘super dominant LAD’ or ‘hyper-dominant LAD’, or ‘type IV LAD’ and it can be considered as an anomalous origin [11,12].

In a patient with an anomalous PDA arising from the LAD, any occlusion of the LAD can lead to massive infarction, possibly resulting in cardiogenic shock or sudden cardiac death. The infarction will possibly involve the anterior wall, the inferior wall as well as the interventricular septum. It is important for an interventional cardiologist to be vigilant of such anomalies whenever performing a catheterization on those patients who present with large infarction involving both the inferior and anterior walls.

As this is an extremely rare anomaly, there are no specific guidelines for treatment. As with all coronary anomalies, coronary computed tomography angiogram is known to be the best noninvasive test for detection [13]. Early revascularization may be potentially lifesaving in these patients in the event of occlusion.

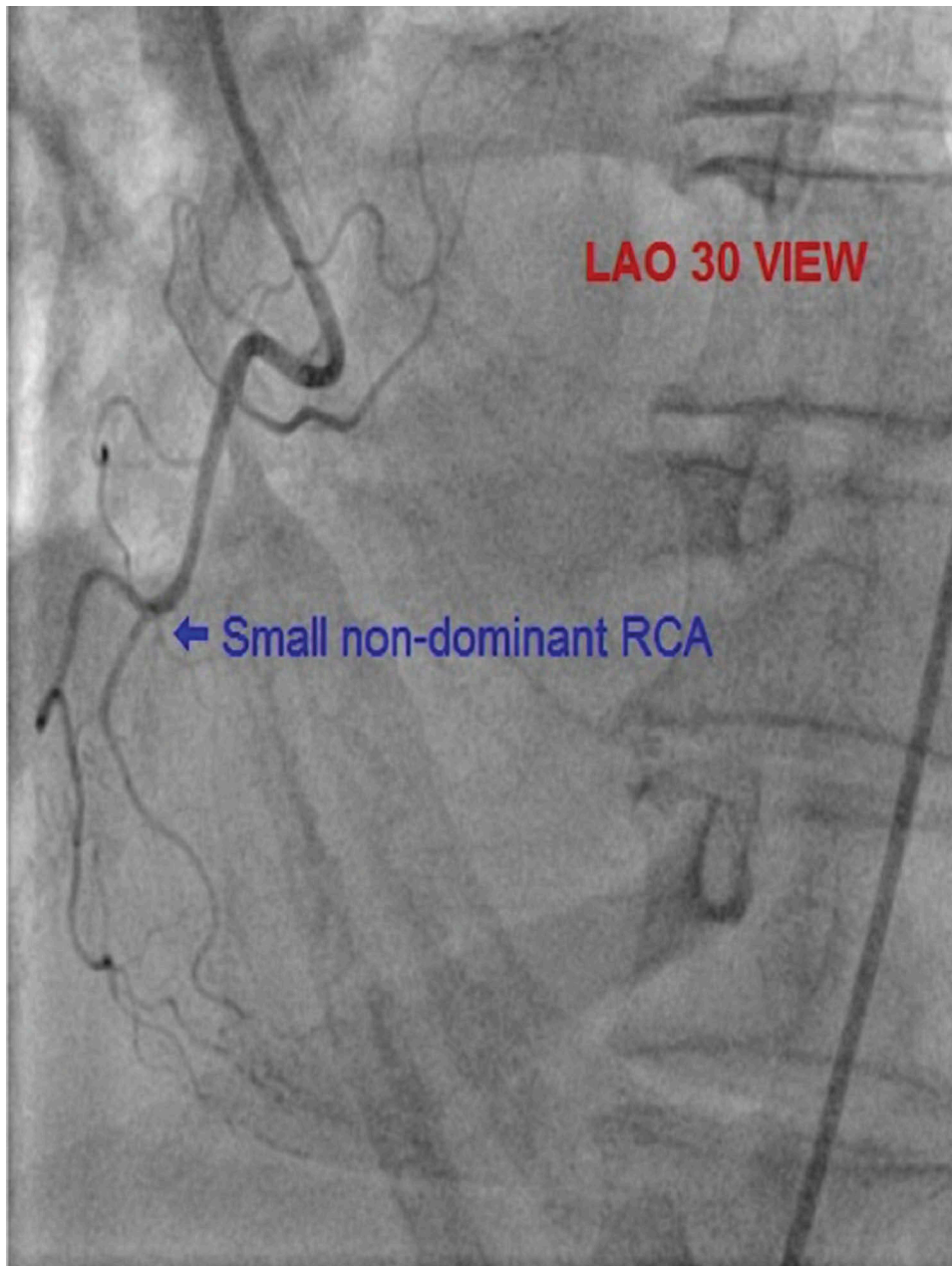


Figure 3. Invasive angiogram identifying non-dominant right coronary artery.

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ORCID

Muhammad Shabbir Rawala  <http://orcid.org/0000-0001-8884-2082>

S. Tahira Shah Naqvi  <http://orcid.org/0000-0002-0920-0039>

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